Type 2 myocardial infarction: incidence, presentation, treatment and outcome in routine clinical practice

Radovanovic, Dragana ; Pilgrim, Thomas ; Seifert, Burkhardt ; Urban, Philip ; Pedrazzini, Giovanni ; Erne, Paul

Abstract: AIMS The clinical definition and optimal treatment for myocardial infarction (MI) type 2 (T2MI) are not well established. We assessed differences in presentation, treatment and outcomes of patients with MI type 1 (T1MI) and T2MI. METHODS The data of MI patients enrolled in the AMIS Plus cohort with T2MI were compared with T1MI using propensity score matching. RESULTS A total of 13 829 patients had T1MI and 1091 (7.3%) T2MI. Patients with T2MI were older, often female, with more risk factors and comorbidities, and less ST segment elevation. After matching for these differences (1091 per group), T2MI patients less often presented with typical chest pain but more frequently with atrial fibrillation (15.6 vs. 4.9%; P < 0.001) and anemia (33.5 vs. 23.3%; P < 0.001) than patients with T1MI. They less frequently received percutaneous coronary interventions (51.1 vs. 76.4%; P < 0.001) and antiplatelet treatment. No differences were found for in-hospital (5.8 vs. 5.6%; OR 1.04; 95% confidence interval 0.72-1.49) and 1-year mortality (11.2 vs. 7.2%; P = 0.38) between matched T2MI and T1MI patients. CONCLUSION Patients who suffered a T2MI had less typical symptoms, were less aggressively treated with anticoagulants, platelet inhibitors or percutaneous coronary intervention, but had similar complications and mortality to those with T1MI. Patients with T2MI are a heterogeneous group that requires further investigation to better define optimal therapeutic approaches.

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Original article

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Aims **The clinical definition and optimal treatment for myocardial infarction (MI) type 2 (T2MI) are not well established. We assessed differences in presentation, treatment and outcomes of patients with MI type 1 (T1MI) and T2MI.**

Methods **The data of MI patients enrolled in the AMIS Plus cohort with T2MI were compared with T1MI using propensity score matching.**

Results **A total of 13 829 patients had T1MI and 1091 (7.3%) T2MI. Patients with T2MI were older, often female, with more risk factors and comorbidities, and less ST segment elevation. After matching for these differences (1091 per group), T2MI patients less often presented with typical chest pain but more frequently with atrial fibrillation (15.6 vs. 4.9%; $P<0.001$) and anemia (33.5 vs. 23.3%; $P<0.001$) than patients with T1MI. They less frequently received percutaneous coronary interventions (51.1 vs. 76.4%; $P<0.001$) and antiplatelet treatment. No differences were found for in-hospital (5.8 vs. 5.6%; OR 1.04; 95% confidence interval 0.72–1.49) and 1-year mortality (11.2 vs. 7.2%; $P=0.38$) between matched T2MI and T1MI patients.**

Conclusion **Patients who suffered a T2MI had less typical symptoms, were less aggressively treated with anticoagulants, platelet inhibitors or percutaneous coronary intervention, but had similar complications and mortality to those with T1MI. Patients with T2MI are a heterogeneous group that requires further investigation to better define optimal therapeutic approaches.**

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Keywords: acute myocardial infarction, outcome, propensity matching, treatment, type 2

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Introduction

The Task Force for the universal definition of myocardial infarction (MI) introduced in 2007 clinically classified MI into five types.[1] The most common form of MI is type 1 (T1MI), which occurs in atherosclerotic coronary arteries by plaque rupture, ulceration, erosion or dissection with thrombotic obstruction. MI type 2 (T2MI) is an imbalance between oxygen demand and supply in the presence or absence of atherosclerotic coronary artery disease. This may be caused by various conditions, such as anemia, arrhythmia, spasm, hypertension or hypotension, but no specific criteria for making a diagnosis of T2MI have been established. Therefore, this type of MI has been the subject of considerable clinical discussion.[2–4] A large spectrum of underlying causes for T2MI open medical management to different treatment strategies.[5,6] The aim of this study was to assess differences in presentation and outcome of patients with T1MI and T2MI, determined by clinicians in 'real world situations' by comparing two main types of MI with regard to baseline characteristics, therapies and outcomes using propensity score matching.

Methods

The AMIS Plus project is an ongoing nationwide prospective cohort of patients admitted with acute coronary syndromes (ACSs) to hospitals in Switzerland. It was founded by the Swiss Societies of Cardiology, Internal Medicine and Intensive Care Medicine in 1997 with the goal to understand the transfer, use and practicability of knowledge gained from randomized trials in the real world of daily clinical practice. Details have been previously published.[7–9]

Among 106 hospitals treating ACS in Switzerland, 83 hospitals temporarily or continuously enrolled patients in AMIS Plus. Participating centers, ranging from community institutions to large tertiary facilities, provided blinded data for each patient through standardized internet-based or paper-based questionnaires. Standard procedures ensured a low percentage of missing data. External monitoring has been regularly performed since 2010 in randomly selected hospitals and patients.

The registry was approved by the Supra-Regional Ethics Committee for Clinical Studies, the Swiss Board for Data Security and all Cantonal Ethics Commissions. Data

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collection is conducted in accordance with the EU Note for Guidance on Good Clinical Practice CPMP/ECH/135/95 and the Declaration of Helsinki.

The AMIS Plus cohort included patients admitted to a participating hospital with one of the following final diagnoses: ST elevation MI (STEMI), non-ST elevation (NSTEMI) or unstable angina. Diagnoses were based on symptoms, ECG and/or cardiac biomarkers, and conformed to the prevailing guidelines in use at the time of inclusion. The universal definitions of MI types were first introduced in 2007.[1] In 2008, the MI types were added to the AMIS Plus case report form. These were then defined according to the discretion of the treating physician.

In this study, we analyzed the patients enrolled in AMIS Plus from January 2009 to July 2015 with final discharge diagnoses of either T1MI of T2MI and compared their presentations, treatments and outcomes.

Information on known risk factors was obtained from the patients' medical history. Dyslipidemia, arterial hypertension and diabetes were considered if the patient was previously treated for such a condition and/or diagnosed by a physician. Patients were defined as obese if the BMI was at least 30 kg/m^2 and as smokers if the patients were smokers at the time of the cardiovascular event. Patient comorbidities were assessed using the Charlson index.[10,11] Immediate drug therapy was defined if administrated within 24 h after admission. Anemia was defined according to WHO criteria [hemoglobin (Hb) below 12 g/dl for women and below 13 g/dl for men] and severe anemia (Hb below 10 g/dl for women and below 11 g/dl for men). Bleeding complications were recorded if deemed clinically relevant by the individual physician in charge of the patient, without the use of a classification system when data collection started. Reinfarction was defined as clinical signs or symptoms of ischemia with ECG changes indicative of new ischemia (new ST changes or new left bundle branch block) and a rerise of biomarkers following the initial infarction. A stroke was defined as any event due to ischemic, thrombotic or hemorrhagic disturbances confirmed by a neurologist or an imaging modality.

The primary outcome measure of the present analysis was in-hospital mortality. Secondary outcome measures were the rates of in-hospital major adverse cardiac or cerebrovascular events defined as a composite of mortality, reinfarction and cerebrovascular events. In a subgroup of patients who gave their informed consent, follow-up was performed at 1 year by means of a standardized telephone interview.

Statistical analysis

We first descriptively analyzed baseline characteristics according to the two MI types. Results are presented as percentages for categorical variables and analyzed using the Pearson chi-square test or Fisher's exact test as appropriate. Continuous normally distributed variables are expressed as means $\pm$ 1 SD and compared using the Student's two-tailed unpaired t test. Continuous non-normally distributed variables are expressed as medians and interquartile ranges and analyzed using the Mann–Whitney U test. In case of missing data, we fused n/N (number of patients with a characteristic/number of patients with available data). A two-sided P value less than 0.05 was considered statistically significant.

To correct for baseline imbalances, we analyzed a propensity matched sample from the crude population. For the computation of the propensity score, the following nine variables were included in a nonparsimonious logistic regression with MI type as the dependent variable: age, sex, STEMI, Killip class more than 2, resuscitation prior to admission, history of diabetes mellitus, arterial hypertension, coronary artery disease and Charlson comorbidity index more than 1. Matching for the subgroup of T2MI and T1MI patients followed 1 year after the event was performed in proportions of 1 : 1, 1 : 5 and 1 : 10 to keep the number of matched patients with T2MI as high as possible. Missing values were replaced by multiple regression imputation for the respective analysis. The validity of logistic regression was assessed using the Hosmer–Lemeshow test. Propensity matching was performed using the package Matching in R.[12]

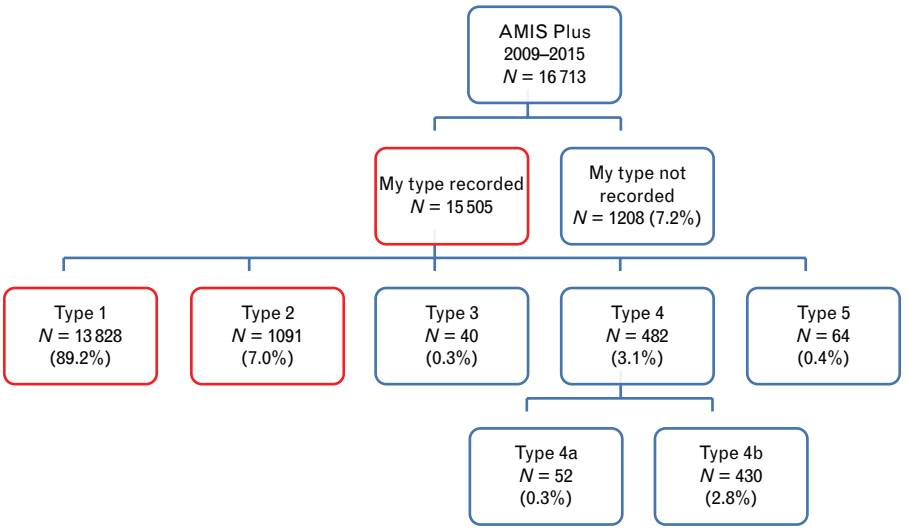
The IBM SPSS Statistics Version 22 (IBM Corp; Armonk, New York, USA) was used for other statistical analyses.

Results

Among 15 505 patients with documented MI types, 14 919 with either T1MI or T2MI were included from 53 Swiss hospitals (Fig. 1). Seventy-four percent were men (aged 64.1 $\pm$ 12.7 years) and 26% were women (aged 71.6 $\pm$ 12.8 years). From these patients, 1091 (7.3%) were classified as having T2MI, and the yearly rate of T2MI patients did not significantly change during the study period ($P = 0.44$). The most frequent clinical presentations thought to be causal to T2MI were known in 685 patients and were anemia 48.3%, atrial fibrillation 24.8%, perioperative 7.4%, hypertonia/hypoxia 5.3%, infection 5.3%, other tachyarrhythmia 5.0%, bleeding 2.5% and other reasons 1.5%.

Table 1 shows the baseline characteristics of patients with T1MI and T2MI in the crude and in the matched samples. Patients with T2MI were older, more frequently women; more frequently had a history of hypertension, coronary artery disease, diabetes and comorbidities; but they less frequently presented with STEMI or with typical chest pain. Atrial fibrillation was present in 15.6% of patients with T2MI vs. 4.9% of T1MI patients, and 33.5% of patients with T2MI had anemia vs. 17.3% of patients with T1MI ($P < 0.001$), and both these differences persisted after matching.

Fig. 1



Clinical classification of different types of myocardial infarction in AMIS Plus 2009–2015.

Immediate drug therapy was different between patients with T1MI and those with T2MI. In the crude sample, 84.5% underwent percutaneous coronary intervention (PCI) compared with 76.4% of the matched patients with T1MI, whereas 15.5%, respectively 23.6%, were treated conservatively. From patients with T2MI, 48.5% received conservative treatment and 51.5% an intervention ($P < 0.001$). From the patients with angiographic data, no angiographic findings [excluding nonobstructive coronary artery disease (<50% stenosis)] were documented for 47/655 (7.2%) patients with T2MI, 31/12 047 (0.3%) in unmatched and in 2/878 (0.2%) in matched patients with T1MI ($P < 0.001$).

In-hospital medical treatment of patients with T1MI and T2MI is summarized in Table 2. Although the administration of beta blockers, angiotensin-converting enzyme inhibitors and angiotensin II receptor antagonist antagonists were comparable between the two groups, patients with T2MI were less aggressively treated with antiplatelet agents and statins.

Odds ratios of in-hospital outcomes of patients with T1MI compared with those with T2MI are shown in Fig. 2 (unmatched) and in Fig. 3 (matched cohort). In the matched cohort, there were no significant differences between patients presenting with T1MI or T2MI with regard to the risk of cardiogenic shock, reinfarction, cerebrovascular events, acute renal failure, bleeding or in-hospital mortality.

Cause of death in both MI types was mainly cardiac: 3.1% in T2MI, 3.0% in T1MI unmatched and 3.5% in matched T1MI patients ($P = 0.87$ and 0.72, respectively).

For subgroup analysis 1 year after discharge, a total of 3375 patients were followed up (3.7% had T2MI and 95.3% had T1MI). No differences were found in rehospitalization rates (24.5 vs. 24.8%; $P = 1.00$) or reinterventions (14.4 vs. 16.2%; $P = 0.70$), but unadjusted mortality was higher in patients with T2MI (11.2 vs. 3.6%; $P < 0.001$). However, mortality of patients with T2MI 1 year after discharge was not significantly different compared with T1MI patients in the 1:10 matched sample (120:1200 patients) (9.2 vs. 6.6%; $P = 0.30$), in the 1:5 matched sample (124:620 patients) (11.3 vs. 7.6%; $P = 0.21$) as well as in the 1:1 matched sample (125:125 patients) (11.2 vs. 7.2%; $P = 0.38$). Among patients with known cause of death after 1 year, 38% of T2MI patients died of noncardiac causes vs. 17% of T1MI patients.

Discussion

The current study documents significant differences between patients classified as having T2MI or T1MI in current everyday practice. Patients with T2MI more frequently had atrial fibrillation, anemia, NSTEMI and fewer treatable stenoses. However, the practical impact of distinguishing between MI types 1 and 2 does not seem to be clearly established in routine care.

In a Danish study, the frequency of T2MI was 26%, in which features of this type of MI were investigated using novel developed criteria in consecutive patients admitted to one university hospital during 1 year[13] and was therefore much higher than in our cohort (7%). It is possible that T2MI was overlooked in our population or not even included in the cohort due to minimal elevation of

Table 1 Baseline characteristics of AMI patients according to myocardial infarction types 1 or 2 ($n = 14\,919$)

	Unmatched			Matched[a]		
Number of patients	MI type 1 13 828	MI type 2 1091	*P*	MI type 1 1091	MI type 2 1091	*P*
Sex female (%)	3517/13 828 (25.4)	387/1091 (35.5)	<0.001	389/1091 (35.7)	387/1091 (35.5)	0.96
Age in years, mean (SD)	65.7 (13.1)	70.8 (13.3)	<0.001	70.6 (12.3)	70.8 (13.3)	0.78
ST segment elevation (%)	7436/13 828 (53.8)	213/1091 (19.5)	<0.001	216/1091 (19.8)	213/1091 (19.5)	0.91
Resuscitation prior admission (%)	811/13 826 (5.9)	40/1091 (3.7)	0.002	39/1090 (3.6)	40/1091 (3.7)	1.00
Killip classes 3/4 (%)	1016/13 783 (7.4)	83/1086 (7.6)	0.72	86/1088 (7.9)	83/1086 (7.6)	0.87
History of hypertension (%)	8009/13 016 (61.5)	766/1042 (73.5)	<0.001	779/1053 (74.0)	766/1042 (73.5)	0.84
Coronary artery disease (%)	3685/13 369 (27.6)	392/1066 (36.8)	<0.001	394/1054 (37.4)	392/1066 (36.8)	0.79
Diabetes (%)	2626/13 137 (20.0)	276/1055 (26.2)	<0.001	312/1047 (29.8)	276/1055 (26.2)	0.065
Charlson Index >1(%)[b]	2875/13 828 (20.8)	400/1091 (36.7)	<0.001	416/1091 (38.1)	400/1091 (36.7)	0.51
Symptoms at admission						
Pain (%)	12 406/13 356 (92.9)	809/1035 (78.2)	<0.001	936/1053 (88.9)	809/1035 (78.2)	<0.001
Dyspnea (%)	3916/12 018 (32.6)	428/969 (44.2)	<0.001	372/960 (38.8)	428/969 (44.2)	0.016
Vital signs at admission mean (SD)						
SBP (mmHg)	137 (29)	136 (29)	0.37	141 (28)	136 (29)	<0.001
DBP (mmHg)	80 (17)	77 (17)	<0.001	79 (17)	77 (18)	0.003
Heart rate (beats/min)	78 (19)	84 (25)	<0.001	79 (19)	84 (25)	<0.001
Heart rhythm						
Sinus rhythm (%)	12 629/13 826 (91.3)	857/1091 (78.6)	<0.001	991/1091 (90.8)	857/1091 (78.6)	<0.001
Atrial fibrillation (%)	566/13 826 (4.1)	170/1091 (15.6)	<0.001	53/1091 (4.9)	170/1091 (15.6)	<0.001
Risk factors						
Family history (%)	3699/11 215 (33.0)	243/824 (29.5)	0.042	274/856 (32.0)	243/824 (29.5)	0.27
Smoking (%)	4978/12 086 (41.2)	282/904 (31.2)	<0.001	296/926 (32.0)	282/904 (31.2)	0.73
Dyslipidemia (%)	7104/12 157 (58.4)	565/977 (57.8)	0.71	625/979 (63.8)	565/977 (57.8)	0.007
Obesity (BMI > 30) (%)[c]	2718/12 173 (22.3)	213/944 (22.6)	0.87	240/945 (25.4)	213/944 (22.6)	0.16
Anemia (%)	2234/12 890 (17.3)	331/987 (33.5)	<0.001	238/1021 (23.3)	331/987 (33.5)	<0.001
Severe anemia (%)	530/12 890 (4.1)	146/987 (14.8)	<0.001	57/1021 (5.6)	146/987 (14.8)	<0.001
Comorbidities						
Heart failure (%)	288/13 441 (2.1)	73/1074 (6.8)	<0.001	51/1066 (4.8)	73/1074 (6.8)	0.052
Cerebrovascular disease (%)	746/13 441 (5.6)	83/1074 (7.7)	0.004	113/1066 (10.6)	83/1074 (7.7)	0.024
Peripheral arterial disease (%)	634/13 441 (4.7)	101/1074 (9.4)	<0.001	80/1056 (7.5)	101/1074 (9.4)	0.12
Moderate-to-severe renal disease (%)	953/13 442 (7.1)	156/1074 (14.5)	<0.001	139/1066 (13.0)	156/1074 (14.5)	0.35
Cancer disease (%)	759/13 441 (5.6)	93/1074 (8.7)	<0.001	82/1066 (7.7)	93/1074 (8.7)	0.43
Dementia (%)	212/13 441 (1.6)	37/1074 (3.4)	<0.01	29/1066 (2.7)	37/1074 (3.4)	0.38

In case of missing data, N = number of patients with available data and n/N = number of patients with a characteristic/number of patients with available data. [a] Matched for age, gender, ST elevation, resuscitation prior admission, Killip class more than 2, history of hypertension, coronary artery disease, diabetes and Charlson comorbidity index more than 1. [b] Charlson Comorbidity Index.

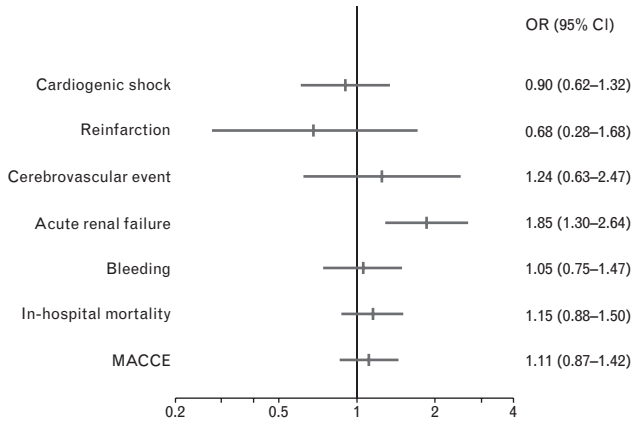
biomarkers or a nonspecific ECG together with an absence of typical chest pain, which could be considered as not fulfilling the AMIS Plus inclusion criteria. The reported incidence of T2MI in the literature varies widely (1.6–26%), reflecting the lack of well defined diagnostic criteria.[6,14]

Table 2 Immediate therapy of AMI patients according to myocardial infarction types 1 or 2 ($n = 14\,919$)

	Unmatched			Matched		
Number of patients	MI type 1 13 828	MI type 2 1091	*P*	MI type 1	MI type 2	*P*
Medicaments						
Aspirin (%)	13 270/13 772 (96.4)	983/1084 (90.7)	<0.001	1034/1089 (94.9)	983/1084 (90.7)	<0.001
P2Y12 inhibitors (%)	12 488/13 769 (90.7)	786/1082 (72.6)	<0.001	944/1087 (86.8)	786/1082 (72.6)	<0.001
GP IIb/IIIa antagonist (%)	2176/13 518 (16.1)	34/1059 (3.2)	<0.001	92/1064 (8.6)	34/1059 (3.2)	<0.001
Heparin (%)	11 774/13 703 (85.9)	801/1082 (74.0)	<0.001	882/1079 (81.7)	801/1082 (74.0)	<0.001
Beta-blocker (%)	7396/13 655 (54.2)	595/1074 (55.4)	0.45	604/1075 (56.2)	595/1074 (55.4)	0.73
ACEI/ARB antagonist (%)	7448/13 665 (54.5)	566/1081 (52.4)	0.17	574/1080 (53.1)	566/1081 (52.4)	0.73
Ca-channel blocker (%)	1244/13 490 (9.2)	161/1066 (15.1)	<0.001	144/1068 (13.5)	161/1066 (15.1)	0.29
Nitrate (%)	6191/13 542 (45.7)	425/1070 (39.7)	<0.001	493/1063 (46.4)	425/1070 (39.7)	0.002
Diuretic (%)	2648/13 532 (19.6)	373/1073 (34.8)	<0.001	308/1066 (28.9)	373/1073 (34.8)	0.004
Statin (%)	10 555/13 689 (77.1)	698/1077 (64.8)	<0.001	819/1082 (75.7)	698/1077 (64.8)	<0.001
Intervention						
Coronary angiography (%)	12 067/13 828 (87.3%)	660/1091 (60.5%)	<0.001	880/1091 (80.7)	660/1091 (60.5)	<0.001
PCI (%)	11 684/13 828 (84.5)	557/1091 (51.1)	<0.001	833/1091 (76.4)	557/1091 (51.1)	<0.001

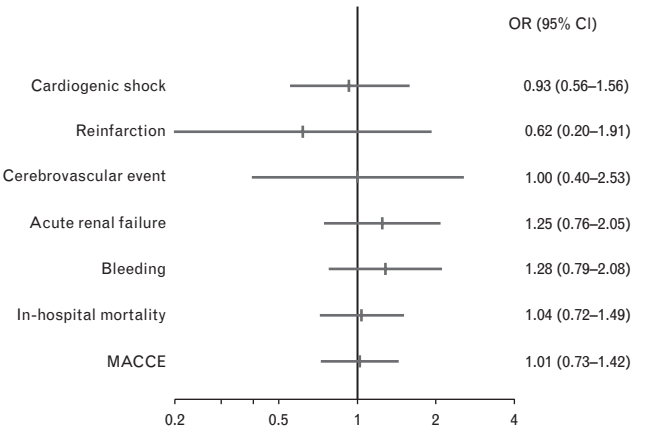
In case of missing data, N = number of patients with available data and n/N = number of patients with a characteristic/number of patients with available data. ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotension II receptor antagonist; P2Y12 inhibitors, clopidogrel, prasugrel or ticagrelor; PCI, percutaneous coronary intervention.

Fig. 2



Odds ratios with 95% confidence intervals of complications and outcomes in a crude cohort of patients with myocardial infarction type 2 vs. patients with myocardial infarction type 1.

Fig. 3



Odds ratios with 95% confidence intervals of complications and outcomes in a propensity matched cohort of patients with myocardial infarction type 2 vs. patients with myocardial infarction type 1.

The question is how to deal with the classification of T2MI in a real-world situation when there is a lack of internationally accepted criteria for diagnosing T2MI. The criteria developed by Saaby *et al.*[13] allow more precise decisions but implementation is difficult in daily clinical practice as the hospitals vary as do the wards. Patients with T2MI had lower peak cardiac troponin (cTn),[13] and although all patients included in our study had biomarker determinations, we were unable to compare this due to multicenter participation of various hospital types with different assays and different reference values. It could be postulated that by using sensitive cTn assays more often, more patients would be classified as having MI2. However, although high-sensitive troponin has been increasingly used, the percentage of patients with MI2 did not constantly increase in our population.

It has been shown that patients with T2MI did not have atherosclerotic plaque rupture compared with patients with T1MI who seldom had normal coronary angiographies.[5] Our unmatched and matched cohort of T1MI patients – 0.3 and 0.2%, respectively – presented with no significant coronary stenosis at angiography compared with 7.2% of patients with T2MI. In the Swedish study, 42.4% of patients with T2MI had normal coronary arteries or nonobstructive coronary artery disease (<50% stenosis).[14] The main difference between these two cohort real-life studies is probably due to the different definitions of angiographic findings.

A T2MI occurs secondary to an ischemic imbalance triggered by a condition such as spasm, arrhythmia, anemia, hypertension or hypotension.[15] In the clinical setting, it is sometimes difficult to estimate the true impact of any of these conditions. For example, it is often not known whether ischemia precipitated the atrial fibrillation or atrial fibrillation precipitated the ischemia.[5] Even severe vasospasm is difficult to diagnose and treat,[16] and distinguishing T1MI from T2MI, particularly in the perioperative period after noncardiac surgery, is often challenging.[5] In a retrospective review of the medical records of 107 patients with T2MI, the most common causes were sepsis, anemia and atrial fibrillation.[17] In this study, the presence of identifiable provoking factors was known for 63% patients with T2MI, and the most frequent clinical presentations thought to be causal were anemia, atrial fibrillation and perioperative status. We have already showed that patients suffering AMI when already in hospital for other reasons had a worse prognosis than those with outpatient-onset AMI.[8]

As yet, there is no consensus on the treatment of patients with a presumptive diagnosis of T2MI. Given the physiopathology, aggressive anticoagulation, platelet inhibition and early PCI could potentially be harmful, and the first line of treatment should often be limited to the underlying conditions.[15] In this study, most T2MI patients with coronary angiography underwent PCI. It could be assumed that the operator must have considered that he/she was dealing with stable coronary disease with an additional trigger that led to MI. Our data confirm that many Swiss hospitals appear to have adopted this approach, as the choice of treatment is determined by the type of MI. It should be recognized that ICD10 codes are insufficient to clearly determine the MI types,[2,18] and this probably has major implications not only for health service research but also for quality and performance measures of hospitals using medical records.

Complications in hospital were similar for both T1MI and T2MI patients except for acute renal failure, which occurred more frequently in patients with T2MI. In-

hospital mortality was not significantly different between T1MI and T2MI patients (unmatched and matched). Unadjusted 1-year mortality was higher in T2MI patients, but adjusted mortality was similar to that of T1MI patients, suggesting that baseline differences rather than type of MI were the main drivers of longer term mortality. Similarly, crude 1-year all-cause mortality in the Swedish study (used merging with the National Population Registry) was much higher (24.7% for T2MI vs. 13.5% for T1MI), but the difference also disappeared after adjustment for baseline characteristics.[14] In contrast, the results from one single-center study in Denmark reported mortality of 119 patients with T2MI as high as 50% after 2 years.[19] This high mortality could be at least partially explained by older age (75 years) and the higher rate of comorbidities (heart failure 22%, prior stroke 20%, chronic obstructive pulmonary disease 26% and diabetes 24%) of these unselected hospital cohort patients.

Limitations

Our study should be considered under some clear limitations. First, this is an observational study, and the classification of MI type was based solely on the treating physician's judgment, which was without external validation and could often have been an educated guess. Second, in the data collection, there was not complete information on the possible precipitating causes of type 2 MI. Furthermore, the rate of T2MI (e.g. perioperative MI) could be underestimated due to treatment of these patients in clinical departments other than cardiology, and therefore they were not enrolled in AMIS Plus. However, there are no clear diagnostic criteria for T2MI, and we believe that the large number of patients included in this cohort study should contribute to improve our understanding of the use of the MI type classification and set the stage for better defining the treatment of patients with T2MI.

Finally, the number of patients with 1-year follow-up data is relatively small and may not be fully representative of the entire cohort. However, there are only few data in the literature on long-term outcomes of patients with T2MI. Our data may therefore contribute to a better understanding and may serve as an incentive for further studies.

Conclusion

Our data document significant differences in clinical presentation, baseline characteristics, treatment and both early and late outcomes between patients with T2MI and T1MI in routine clinical practice. Patients with T2MI presented less frequently with typical symptoms, but often with atrial fibrillation and anemia, and were less frequently aggressively treated with anticoagulants, platelet inhibition or PCI. After correcting for baseline characteristics, complications and mortality in hospital and 1 year after discharge were similar for patients with T1MI and T2MI. Patients with type 2 MI require further investigation to better define it and achieve a consensus on optimal therapeutic approaches.

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Conflicts of interest

There are no conflicts of interest.

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